Spinal Tophaceous Gout

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Gout is a common arthritis caused by the deposition of monosodium urate crystals within joints after chronic hyperuricemia (1). It affects 1-2% of adults in developed countries, where it is the most common inflammatory arthritis in elderly men. Epidemiological data have shown a rise in the prevalence of gout. Gout is the best understood and most manageable of all common systemic rheumatic diseases. However, Neogi et al have reported that it is frequently inappropriately managed (2).

The prevalence of gout is much higher in men than in women. In women, it mainly develops after menopause. The prevalence of gout in men increases with a high consumption of meat, seafood, and fructose, and intake of beer and spirits (1). In addition, rates of gout are heightened with an elevated body-mass index (BMI), but fall with weight loss.

Gout passes through three stages throughout its natural history: I. asymptomatic hyperuricemia, II. episodes of acute gouty arthritis separated by asymptomatic intervals (termed intercritical or interval gout), and III. chronic gouty arthritis, the period when tophi (tophus [L. “porous stone”]) often become apparent (3). Chronic hyperuricemia is the most important risk factor for gout. The risk of acute gout rises with urate concentration. However, many people with a high serum urate level never develop gout.

Acute gouty arthritis most often begins with one joint affected in the lower limbs, usually the first metatarsophalangeal joint, classically termed ‘podagra’ ([Gr. podo food + Gr. agra seizure]). Proinflammatory cytokines play an important role in the inflammation of acute gouty arthritis. Recent clinical trials with IL-1β blockade have revealed an impressive and sustained reduction in patients with recurrent attacks of gouty arthritis (4, 5). Some patients with gout are normouricemic at the time of acute attack, a phenomenon related to a consequence of IL-6 production by the acute inflammatory process (6, 7). We have also demonstrated that there is a tendency for an elevated concentration of IL-17 in synovial fluids from patients with acute gouty arthritis (8).

When left untreated, acute attacks of gout can lead to chronic gout, characterized by chronic destructive polyarticular involvement with low-grade joint inflammation, joint deformity, and tophi (1). Tophi are monosodium urate crystals surrounded by chronic mononuclear and giant-cell reactions. Tophaceous gout develops within 5 years of the onset of gout in 30% of untreated patients (1). Tophi can occur in a variety of locations; Tophi are frequently seen in the helix of the ear, over the olecranon processes, on the Achilles tendons, within and around the toe or finger joints, around the knees, and within the pre-patellar bursae (2).

Tophi deposition can happen anywhere in the body and it sometimes leads to unusual features (1). Spinal involvement can lead to nerve root or cord compression; Barrett et al reported a case with tophaceous gout of the lumbar spine mimicking epidural infection (9), and showed only 32 previous cases with gout arthritis in the spine reported before 2001 (9). In addition, Nakajima et al reported a gout patient with massive spinal tophi in the lumbar spine mimicking a spinal tumor in the absence of any neurological disturbance (10). St. George et al reported a patient with T1-2 spinal cord compression by a degenerate tophus showing a neurological complication (11).

In this issue of Internal Medicine, Yamamoto et al (12) report a 58-year-old woman with a three-year progressive history of chronic arthritis, who had become disabled due to general malaise and fever. Finally, she was diagnosed as having tophaceous gout with uric crystalline by arthrocentesis of the elbow. In addition, the CT of the patient’s cervical spine revealed significant bone erosion and destruction, indicating tophaceous gout in the cervical spine (12). She was successfully treated with prednisolone and urate-lowering therapy. This case is rare; only 16 cases have ever been reported as Yamamoto et al show in Table 2 (12).

Symptomatic gouty arthropathy of the spine is rare. In addition, it is noteworthy that tophaceous gout of the spine may occur in a patient with no peripheral tophi (13). Therefore, tophaceous gout of the spine should be included in the differential diagnosis of patients with symptoms related to
nerve or spinal cord compression, who are known to have or are suspected of having gout.

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References